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HYDROGEN SULFIDE HEALTH EFFECTS AND RECOMMENDED AIR QUALITY STANDARD

ILLINOIS INSTITUTE FOR ENVIRONMENTAL QUALITY

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Hydrogen sulfide is a colorless poisonous gas which is known to cause irritation to the eyes and the respiratory tract and deleterious effects in the nervous system of humans exposed to it. The gas, which has a characteristic "rotten egg" odor at low concentrations, is produced in a variety of industrial and biological treatment processes. Because of the known adverse health effects and the intermittent presence of this gas in Illinois communities, the Environmental Health Resource Center recommends a standard for gaseous hydrogen sulfide of 0.015 mg/m^3 (0.01ppm) based on 8 -hour average sample.

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FOREWORD

The Illinois Pollution Control Board asked the Institute for Environmental Quality to conduct studies whose object is to propose air quality standards for pollutants for which no standards now exist.

This publication is an ingredient of the information required to formulate regulations on which the Pollution Control Board can hold public hearings. Other Institute studies will provide supplemental information required to develop sound regulatory proposals.

This publication deals with hydrogen sulfide. The Institute's Environmental Health Resource Center has prepared the material based on literature searches, investigations, and critical reviews. Recommendations for further research are made, both as a guide for research agencies, and as evidence of the state-of-the-art with respect to hydrogen sulfide.

The Environmental Health Resource Center is a facility of State government established by the Institute for Environmental Quality. Its services are available to anyone interested in environmental health problems.

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The Environmental Health Resource Center is an arm of the Institute for Environmental Quality, managed under contract with the Institute by the University of Illinois, School of Public Health at Chicago, Illinois.

NOTE: The EHRC also appreciates the efforts of John Cantieri, Felix Chen and Anneke Neal in developing this document.

TABLE OF CONTENTS

STANDARD 1

RATIONALE 1

RECOMMENDATIONS 2

SUMMARY 5

Atmospheric Hydrogen Sulfide 5

Natural Occurrence 5

Production Sources 5

Atmospheric Episodes 5

Health Effects of Exposure to Varying Levels of H_2S 7

Acute Poisoning 7

Subacute Poisoning 13

Chronic Poisoning 14

High-Risk Populations 16

Pharmacology of Hydrogen Sulfide 17

Environmental Standard 19

REFERENCES 20

APPENDIX I 25

HYDROGEN SULFIDE HEALTH EFFECTS AND RECOMMENDED AIR QUALITY STANDARD

STANDARD

Hydrogen sulfide (H_2S) is a colorless poisonous gas which is known to cause irritation to the eyes and the respiratory tract and deleterious effects in the nervous system of humans exposed to it. The gas, which has a characteristic "rotten egg" odor at low concentrations, is produced in a variety of industrial and biological treatment processes. Because of the known adverse health effects and the intermittent presence of this gas in Illinois communities, the Environmental Health Resource Center recommends a standard for gaseous hydrogen sulfide of .015 mg/m^3 (0.01 ppm) based on an 8-hour average sample.

RATIONALE

Symptoms associated with human exposure to H_2S include irritation of the respiratory tract and eyes, dyspnea (labored breathing), neurobehavioral inadequacy, and finally, respiratory paralysis and death at high concentrations. These symptoms have been observed in a number of episodic exposures to H_2S. The severest of these episodes occurred in Poza Rica, Mexico, in 1950, where 22 deaths and 320 hospitalizations resulted after an exposure duration of less than 30 minutes. The common complaints in this episode, as well as those in Terre Haute, Indiana (1964) and in Alton, Illinois (1973), included loss of sleep, dyspnea, nausea, headache, and general property damage.

Table II of this report summarizes health effects resulting from occupational and environmental exposures to H_2S. The data indicate that respiratory tract irritation exists at H_2S concentrations of 70 mg/m^3; eye effects, as reflected in increased incidences of decreased corneal reflex, have been observed at 1.0 mg/m^3; and increased incidence of certain subjective complaints, such as nausea and loss of appetite and fatigue, have been reported down to 0.12 mg/m^3.

Certain individuals, because they are afflicted with a particular disease, metabolic disorder or psychological disturbance, have an enhanced susceptibility to exposure to hydrogen sulfide. Included among these high-risk categories are infants, those with eye and respiratory tract ailments, anemia, alcoholism, schizoid or paranoid tendencies, and those who have been previously exposed to H_2S.

On the basis of reported environmental episodes of H_2S exposure, the known health hazards of H_2S, and the existence of hypersusceptibles at risk in the general population, the EHRC recommends a standard of air quality for H_2S at 0.015 mg/m^3. The standard, however, does not prevent the human detection of this gas at considerably lower concentrations, as the odor threshold for H_2S has been reported to be as low as 0.00047 ppm[18] (0.00065 mg/m^3). A standard based on odor consideration is likely to be much lower than the one proposed in this document. In addition, no account has been taken of phytotoxic, animal or material effects. However, based on presently available health data the EHRC believes that the proposed standard will prevent adverse health effects from H_2S exposure in the general and high-risk populations.

RECOMMENDATIONS

The EHRC believes that a standard should be set at the present time for H_2S. However, the following recommendations for future research are proposed to better understand the health effects of this toxic gas.

1. Because of the limited informational data base on the health effects of chronic exposure to low concentrations of H_2S, it appears reasonable to recommend carefully designed epidemiologic surveillance studies (lasting at least one year) that:
   (a) involve more than one area with similar study populations and measurement of health indicators
   (b) employ a standard reliable method of monitoring ambient H_2S concentrations
   (c) comprehensively identify point sources of H_2S release into the atmosphere
   (d) record the proximity of human populations and their high-risk individuals to those point sources

(e) where symptomatology is observed, monitor the atmosphere for H_2S concentrations at increasing radial distances from the point source(s) until an area is reached where citizen complaints are not evident

(f) monitor health effects while short-term elevations in ambient air pollution actually occur rather than determining the effects after the fact

(g) monitor survey populations containing a substantial number of high-risk subjects and, if applicable, people occupationally exposed within the source of H_2S emission

(h) employ a wide variety of thorough techniques (regular telephone contacts, adequate medical examinations, questionnaires, health diaries, vital statistics, etc.) to obtain information on the human health effects of H_2S with careful consideration of subjective sumptoms

(i) publicly encourage citizens to report to local health agencies unusual symptoms from suspected pollutant exposures in an effort to obtain information beyond the selected survey populations

(j) would also attempt to investigate the possible synergistic health effects of H_2S with other materials (CO).

2. Because of the uncertainty of the exact mechanism of effects from H_2S intoxication, further research into the pharmacology of H_2S is essential. This is necessary for the further identification of high susceptibility groups.

3. Because detoxification of H_2S to sulfate or thiosulfates occurs readily in the bloodstream of organisms exposed to H_2S, a research effort to investigate the toxicology of these products appears necessary.

4 Carefully conducted animal studies should be carried out to determine more accurately the health effects of chronic H_2S exposure. The relationships between neurasthenic symptoms and the toxic action of H_2S should be investigated.

5. A study of the possible additive or synergistic health effects of H_2S with other materials, e.g., CO, particularly at low concentrations, is encouraged.
6. Possible sub-clinical effects of H_2S exposure, along with development of tests and procedures to characterize such effects should be investigated.
7. Further work into the identification of population groups particularly susceptible to H_2S is needed, as well as analyses of these populations to determine their reaction to various ambient levels of H_2S.
8. As part of a prevention program, it appears necessary to promote public understanding of H_2S hazards and procedures which can be followed to overcome H_2S exposures.

ENVIRONMENTAL HYDROGEN SULFIDE SUMMARY

ATMOSPHERIC HYDROGEN SULFIDE

Natural Occurrence

Hydrogen sulfide is produced in nature primarily through decomposition of proteinaceous material (animal and vegetable) by bacteria. It develops principally in stagnant water such as found in swamps and polluted water. H_2S also occurs naturally as a constituent of natural gas, petroleum sulfur deposits and numerous volcanic gases and sulfur springs. Data on background air concentration of H_2S due to natural sources are scarce. However, it has been estimated to be between .00015 and .00046 mg/m^3.[1]

Production Sources

Hydrogen sulfide is produced as a by-product in many industrial processes. Production sources include the petroleum industry (refineries and natural gas plants), petrochemical plant complexes, coke oven plants, kraft paper mills, chemical processing industries, dye manufacture, viscose rayon manufacture, sulfur production, manufacture of sulfur-containing chemicals, iron and metal smelters, foundries, food processing plants and tanneries.

The State of Illinois contains many of these possible sources of hydrogen sulfide pollution. A contracted study initiated by the Illinois Institute for Environmental Quality measured ambient H_2S concentrations in both industrial and residential locations. Table I includes the site at which the sampler was located, the location of the sampler on the premises, the suspected source of H_2S emission, and the average and 8-hour maximum concentration values.[7] As indicated in the table, the average 8 -hour concentrations varied from .000188 to .000701 mg/m^3. The highest 8-hour concentration was .0062 mg/m^3.

Atmospheric Episodes

The most serious episode reported involving hydrogen sulfide air pollution occurred in Poza Rica, Mexico on November 24, 1950.[6] There was an accidental release of gas from a hydrogen sulfide absorption unit in a natural gas processing plant. This resulted in the release of considerable amounts of hydrogen sulfide, which quickly spread to residential areas during a time when most people were asleep. The situation was aggravated by an atmospheric temperature inversion with patches of haze and fog. Although the gas was

TABLE I

H_2S MONITORING IN ILLINOIS [1]

Sampler Site	Sampler Location	Possible Source	Concentration[2] in mg/m^3 Average[3]	8-hr. maximum
6047 S. Normandy Chicago (north from plant)	under eave of garage roof	viscose plant	0.000245	0.00088
Route 3 High Street Lockport (east from plant)	under eave of garage roof	oil refinery	0.000418	0.001
106th and Avenue "O" Chicago (east from plant)	under eave of roof	steel company	0.000701	0.0062
Route 2 Herscher (north-northeast from plant)	on roof of tool shed	natural gas plant	0.000188	0.00091
470 Drexel Glencoe		non-industrial site	0.00037	

(1) Data from "Ambient Hydrogen Sulfide Air Sampling Program" conducted by and for the Illinois Institute for Environmental Quality," Project No. 10.015, August 15, 1972.[7]

(2) Accuracy of concentrations is ± 0.05 $\mu g/m^3$.

(3) Average concentration for 20 8-hour samples.

shut off within 20 to 25 minutes, 22 persons died and 320 persons were hospitalized as a result of this brief exposure.

An H_2S episode occurred in neighboring Terre Haute, Indiana in 1964. Biodegradation of industrial wastes in a 36-acre lagoon caused the atmospheric H_2S concentration to reach 0.45 mg/m^3.[5] Of the 81 public complaints, 40 referred to property damage, and 41 referred to health effects. The most frequently reported symptoms were nausea, loss of sleep and abrupt awakening, shortness of breath, and headaches.

Alton, Illinois residents recently experienced an H_2S episode during the summer of 1973. Two hundred and sixty-six of the 458 citizen complaints to the Illinois Environmental Protection Agency concerned health effects, the latter mostly being dyspnea and nausea.[38]

HEALTH EFFECTS OF EXPOSURE TO VARYING LEVELS OF H_2S

H_2S is a toxic and irritant gas whose major effects are exerted on the nervous system, the eyes and the respiratory tract. The type of poisoning--acute, subacute or chronic--depends on the duration and level of exposure to the gas (see Tables II and III).

Acute Poisoning

In the H_2S literature, acute poisoning has, in general, been used to describe episodes of systemic poisoning that have been of rapid onset and in which central nervous system effects have prevailed. Although H_2S is an irritant, the general systemic effects of acute poisoning predominate over the local irritant effects of subacute poisoning.

The threshold for acute poisoning is between 700 mg/m^3 and 1000 mg/m^3.[8,11] Exposure levels of 1400 - 2100 mg/m^3 stimulate the nervous system and are fatal within 30 minutes,[8,17,49] and levels above 2800 mg/m^3 bring instantaneous death by means of nervous system paralysis.[17] The symptoms usually present include sudden fatigue, dizziness and intense anxiety, with paralysis manifesting itself in the immediate loss of olfactory function, collapse and respiratory arrest terminating in death.[40,49] Olfactory paralysis presents dangers as it prevents potential victims from detecting H_2S at concentrations greater than 225 mg/m^3.[13] Cardiac failure usually follows 5-10 minutes after respiratory paralysis. Asphyxiation is indicated as the immediate cause of death. Recovery, if it occurs, is generally rapid and without sequelae.[6,40,41] When sequelae are reported, they are either neurasthenic or otoneurological in character and may include fatigue,

TABLE II
CONCENTRATIONS OF H_2S AND HUMAN HEALTH EFFECTS

Concentration (mg/m^3)	EFFECTS
0.015 (Recommended Standard)	Undefined.
0.12	Increased incidence of mental depression, dizziness and blurred vision. (55)[b]
0.45	Increased incidence of nausea, loss of sleep, shortness of breath, and headaches following chronic exposure. (5)[c]
1.0-10	Increased incidence of decreased corneal[a] reflex (convergence and divergence) after chronic exposure. (27,54)[d]
10-70	Irritation of conjunctiva, fatigue, loss of appetite and insomnia after chronic exposure. (21,24,40)
70-150	Irritation to eyes, i.e., conjunctivitis and keratitis with photophobia, after several hours of exposure. (22,25,26)
150-300	Slight systemic[e] symptoms after many hours of exposure;[a] possible hemorrhage and death within 48 hours.[a] (16,17,49) Irritation to respiratory tract and eyes within one hour, becoming more severe with longer exposure, i.e., conjunctivitis, bronchitis, and keratitis with photophobia. (26,40,49,53)
300-500	Nervous system depression (40); slight systemic symptoms within 4-8 hours;[a] hemorrhage and death within 48 hours.[a] (16,17,49) Irritation to respiratory tract and eyes and loss of sense of smell within 30 min. becoming more severe with longer exposure; photophobia and dyspnea (difficult breathing) within 4 hours. Possible pulmonary edema. (17,29,49)

Concentration (mg/m^3)	Effects
500-700	Slight systemic symptoms within 4 hours;[a] hemorrhage and death within 8 hours. (16,49) Irritation to respiratory tract and eyes and loss of sense of smell within 30 minutes; dyspnea, conjunctivitis, and keratitis with photophobia within one hour. Possible pulmonary edema. (17,29,49)
700-900	Slight systemic symptoms within one hour, i.e., headache, dizziness; unconsciousness and death within 4-8 hours.[a] (16,49) Serious irritation to respiratory tract and eyes (conjunctiva and epithelium) within 30 min, i.e., coughing, bronchitis, pharyngitis, dyspnea, possible pulmonary edema, photophobia, conjunctivitis, and keratitis. (17,29,49)
900-1400	Systemic effects predominate over local irritant effects. Systemic symptoms within 30 minutes;[a] collapse, asphyxia, and death within one hour. (16,49,50)
1400-2800	Systemic effects predominate over local irritant effects. Immediate systemic symptoms, i.e., stimulation of respiration (hyperpnea), followed by respiratory inactivity (apnea);[a] collapse, asphyxia, and death within 30 minutes.[a] (8,17,31)
2800 and above	Systemic effects predominate over local irritant effects. Paralysis of respiratory center;[a] immediate death.[a] (17,35)

(a) Indicates that data was also collected from experiments on laboratory dogs.
(b) Reference 55 refers to a preliminary study in Washington State correlating the high incidence of mental depression with hydrogen sulfide pollution.
(c) Reference 5 reports on the summer H_2S episode in Terre Haute, Indiana.
(d) References 27 and 54 are reports on clinical studies of industrial workers exposed to H_2S in their working environment. Length of employment exposure ranged from 3 months to 26 years.
(e) Systemic effects result from absorption of hydrogen sulfide into the blood stream.[16]

TABLE III

INDUSTRIALLY PRODUCED H_2S EXPOSURES

Concentration	Health Effects & Comments	Ref.
>700-1000 mg/m^3 (Acute)	52 of 89 (58%) workers exposed to H_2S liberated from sewer openings showed signs of intoxication. 12 of the 52 (23%) showed evidence of acute intoxication. 6 of the 12 (50%) lost consciousness but regained it. 3 of the 12 (25%) stopped breathing but were resuscitated. 2 of the 12 (17%) died as a result of respiratory paralysis. 1 of the 12 (8%) developed pulmonary edema.	14
70-700 mg/m^3 (Subacute)	40 of the 52 (77%) were affected less severely and showed the following: predominantly neurological manifestations, dizziness, weakness, nausea, headache, confusion, somnolence, tachycardia, cyanosis, and coughing.	
70-700 mg/m^3 (Subacute)	47 of the 320 hospitalized patients in the Poza Rica episode were closely watched for symptoms and signs. 100% had loss of the sense of smell. 29% complained of a severe headache. 13% experienced conjunctival irritation. 18% suffered from dyspnea. 14% had a persistent cough. 11% recorded vomiting.	6
>700-1000 mg/m^3 (Acute)	During a period from 1943-46, 59 acute poisonings took place at Sweden's shale oil plant. All survived with the help of artificial respiration.	40

Concentration	Health Effects & Comments	Ref.
<700 mg/m^3 (Subacute and Chronic)	Subacute exposures caused 73 cases of conjunctivitis to occur in 1946 and 75 in 1947. 18% of 459 men daily exposed to H_2S complained of irritation to the conjunctiva accompanied by tearing and burning of the eyes. 15% complained of irritation of the respiratory tract. 14% complained of loss of appetite. 12% complained of nausea and GI discomfort. 12% complained of insomnia and headache. 50% complained of fatigue.	40
43 mg/m^3 (Chronic)	332 reported cases of conjunctivitis occurred during the period of December 1-31, 1933 at a viscose rayon plant. (375 mg/m^3 CS_2 also present)	21
33 mg/m^3 (Chronic)	85 reported cases of conjunctivitis occurred during the period of December 1-31, 1934 at the same plant. (290 mg/m^3 CS_2 also present).	
30 mg/m^3 (Chronic)	During 1948, there was an average of 54 cases of conjunctivitis per month in one section of a Belgian viscose rayon plant. (63 mg/m^3 CS_2 also present).	24
20 mg/m^3 (Chronic)	At the same time, there was an average of 13 cases of conjunctivitis per month in a different section of the same plant. (44 mg/m^3 CS_2 also present).	
1.5-8.5 mg/m^3 (Chronic)	16% revealed a decreased corneal reflex following chronic, low-level exposure to H_2S in a Chicago viscose rayon plant. (2-26 mg/m^3 CS_2 also present). 32% complained of fatigue following the same exposure.	27
Low	50% showed a decreased corneal reflex following chronic low level exposure to H_2S in a Pennsylvania viscose rayon plant. (CS_2 also present).	54

gastrointestinal disturbance, convulsion, anxiety and depression, and peripheral acoustic neuritis.

A rather consistent finding on autopsy is the presence of a grayish-green cyanosis externally and a peculiar greenish tint to the blood, brain and viscera. This coloration is particularly evident in the brain.[44,46] H_2S intoxication can produce brain lesions very similar to those resulting from acute anoxia or CO poisoning.[19] Histopathological findings indicate significant cerebral damage to rhesus monkeys exposed for 3 minutes to 700 mg/m^3, including parenchymal necrosis of the cortex, as well as necrosis in the brain stem and one of the basal ganglia, the caudate nucleus. Damage to the brain stem and caudate nucleus could result in the convulsions, rigidity and jactation observed during or after H_2S poisoning. There is presently in the literature no well-established mechanism for the effects produced by H_2S on the nervous system.[17]

Animal studies of acute H_2S exposure have shown H_2S capable of inducing abnormalities in cardiac conduction and pacemaker activities by its inhibitory effect on ATPase and NADPH oxidoreductase, two enzymes essential to intracellular respiration in the myocardial cells.[36,37]

Two distinct mechanisms have been proposed to account for the respiratory failure in acute poisoning:[31]

(1) H_2S at 1400-2800 mg/m^3 induces an active hyperpnea (stimulation of respiration) followed by acute acapnia. This gives way to a period of respiratory inactivity (apnea) which may terminate in asphyxia, unless sufficient CO_2 is reaccumulated to stimulate respiration;

(2) H_2S at 2800 mg/m^3 or above induces immediate paralysis due to a direct action on the respiratory center. According to one source, direct paralysis has occurred at concentrations of 1000 mg/m^3.[51] While breathing may return spontaneously in the first type of failure, in the paralytic form it can only be restored by artificial respiration.

Cases of acute hydrogen sulfide intoxication have been widely reported in the literature[11,29,40,42,43,44,46,47,48,49,50] (see Appendix I, Tables II and III), including acute exposures from industrial accidents involving many people.

Subacute Poisoning

The phenomenon of subacute poisoning is mainly attributed to the direct local irritative action of H_2S on the mucous membranes of the eyes and respiratory tract.[9,17,40,52] Subacute effects are generally experienced within several hours of exposure. The severity of subacute poisoning increases directly with the concentration and duration of H_2S exposure but doesn't terminate in acute poisoning if continued.

Since the eyes are exposed more directly to the surrounding air, they are subject to greater irritation. Thus, irritation of the conjunctiva and/or corneal epithelium is the most common symptom of subacute poisoning. Concentrations from 10-140 mg/m^3 most commonly cause eye irritation, and conjunctivitis has been observed at 20 mg/m^3 and in varying higher concentrations.[11,20,21,22,23,24,40] The most serious consequences to the eyes are:[25,26]

(1) conjunctivitis
(2) keratitis and rupture of the corneal vesicles
(3) temporary loss of vision

Increased humidity is an important contributory factor that increases these irritative effects, which may not appear as severe during the exposure as after due to the anesthetic effect of hydrogen sulfide on the nerves supplying the corneal membrane.[27,28] Symptoms preceding these painful conditions include lacrimation, hyperemia, everted eyelids, retroorbital aching, blepharospasm, distorted and blurred vision, photophobia and rainbow around lights.

Respiratory tract irritation in the form of bronchitis, rhinitis and pharyngitis has occurred after prolonged exposure to 70-280 mg/m^3 of H_2S.[49] These effects have occasionally occurred after 5-10 minutes exposure at 700-840 mg/m^3. Pulmonary edema and pneumonia may follow prolonged exposure to 350-840 mg/m^3. The edema usually does not appear until some time after the exposure, and thus constitutes an important and sometimes fatal sequelae of subacute poisoning.

Recovery from subacute poisoning is often slow, sometimes requiring several days. This prolonged course is due to the inflammatory

processes which follow irritation. Coughing is often persistent for some time following exposure.

The ability of hydrogen sulfide to induce serious respiratory tract irritation is due to its relatively insoluble nature.[17] This property enables the irritative effects of the gas to extend uniformly throughout the entire respiratory tract,[30] so that deeper structures such as the atria and alveoli are involved.[31]

The microscopic pathology of subacute poisoning as observed in animals is much more distinctive than the rather unremarkable findings of acute poisoning. The lungs show evidence of local congestion, and the cells lining the alveoli are filled with transudate and blood.[49] Cases of repeated poisoning also show leukocytes in the alveolar spaces.[49]

The degree of lung damage induced by H_2S is also aggravated by increased humidity.[9,26,32] In addition, H_2S in concentrations greater than 20 mg/m^3 reduces vital lung capacity by 5.3% on the average. This effect appears to involve the autonomic nervous system although, as yet, no precise mechanism has been set forth.[34]

Although subacute poisoning mainly involves local irritation, slight systemic intoxication resulting in headache, fatigue, irritability, insomnia, mild depression, and gastrointestinal disturbances may be caused by exposure to low concentrations of H_2S for several hours.[11]

Chronic Poisoning

Chronic poisoning may be defined as a prolonged state of symptoms or a debilitated condition which results from a single or repeated long-duration exposure to concentrations of H_2S that, in themselves, do not produce symptoms of acute or subacute poisoning. Tables II and III summarize available chronic exposure studies. The occurrence of neurasthenic symptoms which cannot be explained otherwise have usually constituted the meager basis of the diagnosis of chronic H_2S poisoning. A well-documented threshold for chronic poisoning does not exist, and there is wide disagreement concerning the very existence of the pathological state defined as chronic poisoning, since some authors claim that a consistent set of objective symptoms is lacking.

One study has compared the subjective and objective symptoms of two groups of workers exposed chronically to different levels of H_2S: Group I being exposed to concentrations exceeding 28 mg/m^3 and Group II to much lower but unspecified amounts.[40] While objective symptoms such as irritation were not unusually frequent in Group I, neurasthenic symptoms (primarily fatigue) were significantly higher in this group, and their frequency in both groups increased with length of employment and degree of H_2S exposure. The possibility of contributing effects from other gases, however, was not excluded. One must apply caution in evaluating subjective complaints. Since it was not possible to draw firm conclusions from absolute frequencies, the authors based them exclusively on a comparison between the more exposed group I and the less exposed group II.

A second study of chronic intoxication involved workers in a viscose rayon plant, where exposure to 3-40 mg/m^3 CS_2, as well as to 1.5-8.25 mg/m^3 of H_2S, complicates interpretation of data on subjective symptoms. Nevertheless, 32% of the exposed workers complained of fatigue. In addition, 16% were found to have decreased corneal reflexes, an objective sign which is measurable, as opposed to 12% of the control population. Length of employment exposure ranged from 3 months to 17 years.[27]

An older study of workers in the same industry reported 50% of the exposed workers had decreased corneal reflexes when exposed to CS_2 and H_2S in roughly the same range as the previous study.[54]

A recent report from Seattle correlates the high incidence of mental depression on the West Coast with levels of H_2S air pollution. The preliminary study used suicide, divorce rates, and psychiatric admissions to hospitals as indicators of depression and found that the highest rates correlated with the highest hydrogen sulfide distribution. The exposure was chronic and the highest measured H_2S level was 0.12 mg/m^3. Other effects observed in this study included dizziness and blurred vision.[55]

The general picture of chronic poisoning includes local irritation of the eyes and respiratory tract, bradycardia, cold sweats, fatigue,

gastrointestinal disturbances, headache, mental depression, chills and abnormal peripheral reflexes indicative of depression of the nervous system.[56]

HIGH-RISK POPULATIONS

Knowledge of the toxicology of H_2S permits the identification of individuals who, because of some disease process, metabolic disorders or psychological disturbance, cannot tolerate H_2S or its by-products and therefore possess enhanced sensitivity to this gas. In terms of evidence in the literature, however, the identification of "high risk" groups within the population appears similar to the characterization of chronic poisoning, i.e., the evidence is sparse, without stringent controls or reproduced results. Having stated this qualification, we have undertaken to identify those individuals whose capacity to tolerate H_2S is reduced.

First, individuals with eye or respiratory tract problems would seem especially vulnerable to the caustic and irritant, and sometimes fatal, action of the gas at these sites. Conjunctivitis, eye infection, emphysema, TB viral pneumonia and bronchitis would seem to aggravate the irritative property of H_2S in individuals with such problems. Reports of studies investigating sensitivity to H_2S of people with these chronic disabilities are limited.

Second, individuals with anemia would appear to constitute another vulnerable segment, since the detoxification of H_2S relies on the presence of O_2 in the blood, and the amount of O_2 is dependent on both the amount of Hb in the red cells and on the number of red cells. Again, no reports of the increased susceptibility of this group have been found in the literature.

Third, alcoholics or individuals who have consumed alcohol within 24 hours of exposure have been overcome by unusually small concentrations of H_2S indicating that alcohol and H_2S do not mix.[57] In community terms, alcoholics may constitute a hypersusceptible population.

Fourth, persons having psychiatric problems are a poor risk at any H_2S level. Individuals with schizoid or paranoid tendencies become

markedly worse following exposure. Neurotic individuals have developed innumerable bizarre symptoms, many of which remain for a long time as after-effects of the exposure.[57]

Fifth, Russian studies have indicated that growing infants may be particularly sensitive to exposure of H_2S at even very low concentrations. Small concentrations of H_2S (0.1-10 mg/m^3) have produced a symptom complex usually manifested as undernourishment, delayed growth, general weakness and retarded physical and neuro-physical development (e.g., began walking late and teeth were slow in cutting through), as well as assorted gastrointestinal disturbances.[58,59] Whether the general impaired development and poor health were the result of direct toxic action or were secondary to undernourishment and gastrointestinal disturbances induced by the gas was not established. Confirmation of these studies in the West is lacking.

Finally, industrial data indicate that previous exposure to H_2S tends to induce hypersusceptibility and not tolerance to H_2S,[56] although some believe that previous exposure has no effect either way. No scientifically valid studies have been done to demonstrate either claim. Nevertheless, those previously exposed may comprise a hypersusceptible population.

PHARMACOLOGY OF HYDROGEN SULFIDE

The physiological effects of H_2S are dependent upon duration, frequency and intensity of exposure and upon individual susceptibility. The principal route of absorption of H_2S into the bloodstream is through the lungs.[8,9] Although absorption through the skin[8] and intestinal wall[10] have been reported, these routes of entry are insignificant both in terms of the amount absorbed and the consequent toxic effects.[8,9,11]

The detoxification of H_2S, the body's defense mechanism, is dependent upon the oxygen content of the blood.[12,13] Existing only momentarily in the blood as dissolved H_2S,[13] the vast proportion of the gas reacts with oxygenated hemoglobin (HbO_2) to produce such compounds as thiosulfate or sulfate[8], which are eliminated through the kidneys.[8,15]

The present evidence suggests that neither enzyme nor cofactor are important catalysts in this reaction[14] which occurs primarily in the blood[12,13] and, to a minor degree, in muscle where H_2S reacts with myoglobin.[13] The remaining trace amount of unoxidized, dissolved H_2S is eliminated by the lungs.[8,9,12] The detoxification process is very rapid, preventing H_2S from acting as a cumulative poison.[17] This fact is demonstrated by the finding that slow intravenous injection several times the lethal dose of Na_2S has no apparent effect whereas a rapid injection of a lower dosage is fatal.[12,49]

The portion of the gas which first comes into contact with moist tissues such as the eyes and the respiratory mucosa undergoes an additional reaction prior to oxidation by hemoglobin. The gas combines with the alkali in the tissues to form sodium sulfide, a caustic irritant[16] responsible for the irritative effects of H_2S, and the following hydrolysis presumably occurs:[16]

$$Na_2S + 2H_2O \rightleftharpoons H_2S + 2NaOH$$

The H_2S liberated by this reaction is then oxidized as before.

The systemic effects result from absorption of H_2S into the bloodstream at a rate faster than it can be detoxified and the consequent perfusion of the central nervous system by unoxidized H_2S. Various investigations indicate that the systemic effects are attributable to H_2S blocking action on certain enzyme systems, some of which are directly involved in the oxidative processes of cells.[13,37] Respiratory paralysis, as a particularly acute systemic effect, has been attributed by others to the reflexes resulting from the irritative action of the gas on the carotid sinus.[8]

The precise mechanism by which H_2S exerts its toxic properties via enzymic inhibition has not been firmly established, but it is generally agreed that it occurs by the formation of sulfides of numerous cations. This reactivity essentially removes, at whatever degree, enzymic metal cofactors that are necessary for optimal activity. Such inhibitory effects due to the presence of H_2S have been reported for the dehydrogenases (succinic), phosphatases (ATPase), oxidases (DOPA oxidase), carbonic anhydrase, dipeptidase, benzamidase and such iron-containing enzymes

as the catalases, peroxidases and the cytochromes (respiratory enzymes).[13,36,39]

ENVIRONMENTAL STANDARD

The data referenced in Table II indicate that respiratory tract irritation exists at H_2S concentrations of 70 mg/m^3; eye effects, as reflected in increased incidences of decreased corneal reflex, have been observed at 1.0 mg/m^3; and increased incidence of certain subjective complaints, such as nausea and loss of appetite and fatigue, have been reported down to 0.12 mg/m^3. The occupational data available concerning neurasthenic effects and decreased corneal reflex indicated significant increments in both the objective neurological sign and the subjective report.[27,40,54] Unfortunately, the background incidence of the symptoms in the general population is presently unknown.

Since the standard must protect many high risk groups within the general population, the level of .015 mg/m^3 chosen as an environmental standard is considered adequate, with presently available knowledge, to protect the general population against the health hazards of exposure to H_2S.

This standard is based only on consideration of the reported health effects of H_2S. No account was taken of effects of H_2S on plants, materials or animals. H_2S has a very pungent irritating odor (rotten eggs). A standard which would prevent all odor problems is likely to be much more stringent than the one recommended on the basis of health effects. This standard will not solve the odor problems associated with the release of H_2S into the atmosphere.

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APPENDIX I

REPRESENTATIVE SUMMARY OF ACUTE POISONING BY H_2S

1. A thirty-five year old workman collapsed a minute or two after descending into a 15 ft. deep sewer to collect water samples. Two would-be rescuers also collapsed. None of the three responded to vigorous resuscitation. At autopsy all reeked of hydrogen sulfide odor and presented a striking grayish-green cyanosis externally and hemorrhagic pulmonary edema internally, as well as a greenish coloration of the brain and viscera.[46]

2. An episode of severe H_2S pollution occurred in Poza Rica, Mexico in 1950. An accident at the sulfur-recovery unit of a natural gas treatment plant resulted from efforts to increase the rate of flow of gas to the plant's rated capacity. When gas began surging through the unit, unburned H_2S escaped into the atmosphere. The acute exposure of the pollutant caused the death of 22 persons and the hospitalization of 320. The onset, symptoms and signs, and pathology were consistent with H_2S poisoning.[6] .

3. A workman attempting to repair a ruptured outfall line from a poultry feather cooker was found dead. A subsequent autopsy indicated that death was caused by the inhalation of hydrogen sulfide produced in the cooking cycle and discharged through the outfall pipe. Significant findings on autopsy included marked pulmonary edema, dusky cyanotic color of brain and skin, and evidence of cerebral edema.[29]

4. A 42 year old man and his 17 year old son become unconscious while emptying a cesspool in their back yard. The man fell unconscious and when he revived, attempted to move his now unconscious son. He became unconscious again, and when revived by neighbors, made a speedy and full recovery. The boy remained unconscious and died despite continued artificial respiration. Autopsy concluded that the cause of death was asphyxia following H_2S intoxication.[47]

5. A 46 year old sewer worker, investigating an obnoxious odor in a manhole, lost consciousness as he ascended. He remained at the bottom of the manhole for at least 30 minutes before he was brought to the surface. Although revived by artificial respiration, the victim manifested unusual sequelae, including neurological and cardiovascular deficits, the latter involving a right bundle branch block which was picked up following an electrocardiogram.[48]

6. A 48 year old farmer was found unconscious while shovelling manure at a dung heap. After lying there for two hours, he was taken to a hospital, where he had convulsions typical of hydrogen sulfide intoxication. Changes reminiscent of myocardial infarction were found in the electrocardiogram but disappeared in a few days. A month and a half later the patient felt well apart from slight dizziness.[43]

7. A 31 year old refinery worker was found unconscious near the area of a spill of diethanolamine charged with H_2S. One half hour later he was found and was revived with artificial respiration. He developed pulmonary edema, convulsion, and myocardial damage following the exposure. The outcome was favorable, but sequelae in the form of depression and lassitude persisted for many months.[42]

8. Two workers charged with disposing inoperative cylinders of H_2S gas by perforating its side with a heavy-caliber bullet became unconscious when they failed to escape the leaking gas. Both were revived by artificial respiration. No sequelae were present except for a slight numbness in one of the men for a period of three days following the exposure.[11]

9. A 33 year old industrial waste tank driver was seen unconscious next to the tanker which was being worked in a narrow alleyway. The gas which escaped from the tanker on cleaning was later found to be hydrogen sulfide. The worker was not responsive to artificial respiration, and observers commented on the intense cyanosis of the victim.

Pulmonary edema and hemorrhages were found at autopsy, as were green discoloration of many parts of the brain.[50]

10. A number of men were cleaning a condenser box in a refinery preparatory to painting it. When one man noticed H_2S odor, he warned the others, and all immediately started to climb the ladders. Two men collapsed, and rescuers failed to reach them while resuscitation was still possible. Pulmotors and lung motors were used for one hour without success.[49]

11. An industrial accident at a chemical plant resulted in the release of lethal quantities of hydrogen sulfide. Twelve of 89 workers present in the area became severely intoxicated and lost consciousness. Two of the twelve died from respiratory paralysis.[14]